such as well-fitting gloves, masks, and protective eyewear, should be readily available. Consideration should also be given to monitoring the compliance with infection-control policies so that appropriate remedial interventions can be instituted. Supervisors should be responsible for assuring that employees are aware of the hazard and are complying with required work practices.

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Marijuana Testing

THE ANALYSIS of urine to detect drug use is widely used today to protect employee health and safety and to help maintain industrial quality and productivity. Marijuana testing procedures are of concern because the results are more difficult to interpret than results for other abused substances. Urine is currently the specimen of choice, being easier to test than either blood or saliva and, when testing positive, it will remain so for a longer period of time. Urine testing is less invasive, less costly, and has a shorter turnaround time than blood testing.

Marijuana (Δ9-tetrahydrocannabinol [THC]) enters the circulation rapidly by smoking (minutes) and more slowly by ingestion (1½ to 3 hours) and is so highly metabolized that only a small fraction that enters the bloodstream is excreted unchanged in the urine. THC is rapidly converted by hepatic enzymes to several metabolites, the most prevalent of which is THC carboxylic acid. THC is stored mostly in adipose tissue and may accumulate faster than it can be removed in persistent users. For example, in a person using three or more joints per day who stops smoking marijuana completely and then adopts an exercise fitness program, thereby mobilizing body fat, urine specimens will test positive for THC at 50 ng per ml to 100 ng per ml or more for over two months, whereas one who smokes an occasional joint will test positive at from 50 to 100 ng per ml or more for three to four days. In addition, passive inhalation of marijuana smoke by nonusers occasionally will result in a concentration of 20 ng per ml and in rare cases as high as 40 ng per ml. THC testing is divided into sensitive screening tests (presumptive) and specific confirmatory tests (definitive). Most current screening technology involves immunoassay techniques, the private sector using primarily the enzyme-multiplied-immunoassay technique—EMIT-d.a.u. for a large number of specimens (more accurate), and EMIT-st for a small number of specimens (less accurate) (Syva Corporation)—whereas the federal government and military prefer the radioimmunoassay (RIA, Roche). A fluorescent immunoassay technique using polarized light—known as TDx (Abbott Laboratories)—has recently become available.

Abnormal immunoassay results do not necessarily imply the presence of THC, as any other agent in the specimen that binds to the antibody will result in a false-positive test. Also, the EMIT screen may test false-negative if a specimen is adulterated by the addition of bleach, detergent, salt, or vinegar or diluted with water from a tap or a toilet bowl.

Gas chromatography/mass spectrometry (GC/MS) is currently the most reliable confirmatory technique. In terms of analytic accuracy, EMIT-d.a.u. has a specificity in the range of 90% and a sensitivity in the range of 95%. GC/MS has a specificity of greater than 99.9% and a sensitivity of greater than 99.9%. Thus, a combination of screening by EMIT and confirmation by GC/MS will yield virtually 100% accuracy in testing for marijuana.

Marijuana testing as part of a well-designed program for the screening of employees and applicants for substances of abuse may help define the magnitude and seriousness of the problem and protect and improve health and safety in an ethically acceptable manner.

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Occupational Health Implications of a Toxic Spill of Propylene Dichloride

GIVEN THE ENORMOUS VOLUME of potentially hazardous materials being transported, it is inevitable that clinicians will see patients who have been exposed as a result of accidental spills.

On February 5, 1981, a truck containing propylene dichloride leaked 2,000 gallons. Propylene dichloride is a volatile chlorinated hydrocarbon that is a mucous membrane irritant and a central nervous system depressant. As a result, hundreds of people were evacuated from their homes; 129 persons were treated at a nearby emergency department, 15 of these being admitted to hospital. The persons exposed included truck drivers who were also present at the site of the spill, 2 California Highway Patrol officers, 12 firefighters, and a number of hospital employees who were secondarily exposed as a result of contact with the victims' contaminated clothing. A number of those persons had persistent complaints.

Propylene dichloride and closely related materials have been used as soil fumigants for a number of years. Unfortunately, there have been previous incidents involving the accidental release of propylene dichloride or related materials in combination with other agents. Some of those exposed experienced a good deal of chest discomfort, dyspnea, and cough; of this group, some had persistent chest pain or discomfort and fatigue.

Physicians involved in treating workers exposed to such an accident or release face many challenges. A step-by-step approach for the management of these types of exposures has been outlined. Indeed, the emergency department experience of persons in the above-cited accident reinforces the need for advance planning for both examination and treatment. The treatment of patients who have inhaled irritant materials in general focuses on supportive management. This includes the use of oxygen, bronchodilators where important, and, in the view of some authors, the use of systemic steroids to minimize complications.

Hospitals are rarely well prepared to minimize exposure of their own personnel to chemical hazards. The hospital spokesperson in this circumstance indicated that the emergency room personnel wore caps and gowns. Surgical masks will be of minimal, if any, benefit in dealing with possible chemical inhalations. Hospitals might be well advised to have available in their emergency departments a number of respirators designed to protect against exposure to organic vapors. Personnel need to be trained in the use of respiratory protection.

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Reactive Airways Disease Syndrome

THE REACTIVE airways disease syndrome describes the development of airway hyperreactivity and asthmatic symptoms following a single exposure to a high level of pulmonary irritants. The syndrome differs from occupational asthma in several ways: First, it develops only with very high levels of exposure to materials that are directly irritating, whereas occupational asthma develops after repeated exposures to relatively low levels of sensitizing materials. Second, the reactive airways disease syndrome develops after only a single exposure, whereas occupational asthma depends on repeated exposures with sensitization. Third, while patients with occupational asthma have a particular sensitivity to the material causing the syndrome, such as toluene diisocyanate, patients with reactive airways disease are not peculiarly sensitive to the causative agent. Fourth, a large fraction of occupational asthma is caused by allergic sensitization, whereas this is not the case with the reactive airways disease syndrome.

The diagnosis of the syndrome is based on two factors: first, a history of exposure to a high level of a toxic material, and, second, the presence of airway hyperreactivity. Various toxic, nonsensitizing materials have been shown to cause reactive airways disease, such as sulfur dioxide and ammonia. In addition, we have observed its occurrence due to bromine and to products of plastic pyrolysis (burning plastic).

Airway hyperreactivity may produce intermittent dyspnea and wheezing. Although spirometry findings may be normal between exacerbations, airway hyperreactivity can be detected and objectively quantified by tests of airway hyperresponsiveness. Airway function is determined by spirometry (or whole-body plethysmography) as a baseline and after administering a placebo, such as a saline aerosol. Gradually increasing doses of the provocative agent are then inhaled, with airway function measured after each dose. An abnormal response—such as a 20% decline in the forced expiratory volume in one second—will not occur in normal persons. The test is safe to do because it is discontinued after the first abnormal response to the increasing doses; if necessary, an inhaled bronchodilator may be used to reverse the bronchospasm.

Symptoms developing after a heavy exposure in a previously asymptomatic patient suggest reactive airways disease. Treatment should be similar to that for asthma. Workers with the syndrome can have asthmalike symptoms and physiologic impairment years after the incident. The long-term prognosis is uncertain.

Reactive airways disease is specific for high-level exposures; low-level exposure to respiratory irritants has not been shown to produce the syndrome. In summary, the reactive airways disease syndrome is a new name for an old phenomenon. Despite improved industrial hygiene practices, accidental single episodes of high-level exposure unfortunately are still frequent. Physicians should counsel patients after such exposures to report any symptoms suggestive of asthma or respiratory impairment so that appropriate confirmatory testing may be done. In addition, clinicians should carefully question patients with adult-onset asthma about any acute exposure events surrounding the onset of their asthma.

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Passive Smoking and Lung Cancer

ACTIVE CIGARETTE SMOKING has been amply shown to be the major cause of lung cancer. A dose-response relationship is seen with no apparent threshold, consistent with current biologic theory. Side-stream smoke, the smoke released from a burning cigarette tip, contains all the toxic and carcinogenic compounds in mainstream smoke, many of which are found in significantly higher concentrations in side-stream than in mainstream smoke. Studies of nonsmokers have shown that exposure to environmental tobacco smoke results in the uptake of tobacco smoke constituents. Thus, one would predict an increased lung cancer risk from such exposure.

Recent epidemiologic studies have now consistently shown an increased risk of lung cancer among nonsmokers exposed to environmental tobacco smoke. At least 13 studies from 6 countries have been reported, 11 of which found an increased risk of lung cancer among nonsmokers. The magnitude of the risk for the highest exposed groups has generally been in the range of twofold to threefold, but small sample sizes resulted in wide confidence limits for many of the estimates. The data on passive smoking have been reviewed in the Surgeon General's report, which found that "Involuntary smoking is a cause of disease, including lung cancer, in healthy nonsmokers." A recent review by the National Academy of Science also found that "exposure to [environmental tobacco smoke] increases the incidence of lung cancer in nonsmokers." For both of these reports the epidemiologic studies were critically reviewed and the conclusion made that errors in these studies did not negate the observed findings. The average increase in the lung cancer risk for nonsmokers exposed to environmental tobacco smoke is generally felt to be about 30% above the risk for nonsmokers without such exposure, or a relative risk of 1.3. This estimate is consistent with estimates based on low-dose extrapolation of active smoking risk models.

Many of the epidemiologic studies have considered only spousal smoking or home exposure as the source of environ-